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Environmental Tobacco Smoke and Behaviors of Inner-City Children With Asthma

Maria Fagnano, BA, MPH, Kelly M. Conn, MPH, and Jill S. Halterman, MD, MPH

From the Department of Pediatrics, University of Rochester School of Medicine and Dentistry, and the Children's Hospital at Strong, Strong Memorial Hospital, Rochester, NY

Abstract

Objective—To explore the relationship between environmental tobacco smoke (ETS) exposure and behavior among inner-city children with significant asthma.

Methods—We analyzed baseline data for 200 children 4 to 10 years old who were enrolled in an asthma program. Environmental tobacco smoke exposure was measured by the child's salivary cotinine level. Caregivers completed the 28-item Behavior Problem Index (BPI). Positive responses were summed for a total BPI score, and children with scores >14 were considered to have significant behavior problems. We conducted Student *t* tests and multivariate regression analyses to determine the association of children's cotinine levels with BPI scores.

Results—Overall, 56% of children were male, 65% were black, and 72% had Medicaid. Mean cotinine level was 1.47 ng/mL. Overall, 30% of children had total BPI scores >14. Children with cotinine values >1.47 ng/mL had significantly higher scores compared with children with lower cotinine values on total BPI (12.5 vs 10.2), as well as externalizing (9.0 vs 7.2), antisocial (2.3 vs 1.7), and immature (2.1 vs 1.6) subscales. In a multivariate model, log cotinine remained independently associated with externalizing ($P = .04$), headstrong ($P = .04$), and antisocial behavior ($P = .04$).

Conclusions—Cotinine levels are independently associated with problem behaviors among this sample of urban children with asthma.

Keywords

behavior; childhood asthma; environmental smoke exposure; inner-city

Asthma is a leading cause of childhood morbidity,¹ affecting approximately 9 million children in the United States.² The burden of childhood asthma is extensive, including high rates of hospitalizations³ and emergency department visits,^{1,3,4} absenteeism from school and work,³ and impaired quality of life.⁵ Several studies also have linked asthma to childhood behavior problems.^{6–8} One study found that children with severe asthma were 3 times more likely to also have significant behavior problems compared with children without a chronic condition.⁶ Similarly, in a meta-analysis conducted in 2001, children with persistent asthma symptoms had higher levels of behavioral problems compared with healthy children.⁷ In a recent cross-sectional analysis of young inner-city children entering kindergarten, children with persistent asthma symptoms demonstrated behavior problems in both internalizing and externalizing domains compared with children without asthma.⁸

Address correspondence to Maria Fagnano, BA, MPH, University of Rochester School of Medicine, Box 777, Strong Memorial Hospital, 601 Elmwood Ave, Rochester, New York 14642 (e-mail: maria_fagnano@urmc.rochester.edu).

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Among children with asthma, environmental tobacco smoke (ETS) is associated with worsening respiratory symptoms and decreased pulmonary function.³ In the United States, approximately 60% of children aged 3 to 11 years are exposed to secondhand smoke,⁹ and 25% live with one or more smokers in their home.¹⁰ Children from poor and African American backgrounds suffer disproportionately from asthma^{4,11} and are also more likely to have higher levels of ETS exposure.^{10,12,13} One large study found that more than half of inner-city children with asthma live with at least one smoker in their homes.¹⁴

There is increasing evidence that ETS exposure is also associated with higher rates of behavior problems in children.^{15–17} Some studies demonstrate a dose response effect of tobacco smoke exposure on behavior outcomes; with higher levels of smoke exposure associated with higher rates of behavior problems.^{18,19} Animal studies have demonstrated changes in the central nervous system related to both prenatal and postnatal smoke exposure.^{20,21} This association is of particular interest for children with asthma because rates of both ETS exposure and behavior problems are elevated.

The objective of this study was to explore the relationship between salivary cotinine, a biomarker of ETS exposure, and behavior among inner-city children with significant asthma. We hypothesize that children with higher levels of cotinine will have worse behavior scores.

METHODS

Setting and Participants

This study uses baseline data collected from a community-based sample of 3 to 10 year-old children enrolled into a school-based asthma intervention in inner-city Rochester, New York. We identified children through school health forms. A screening form was administered by telephone with the child's primary caregiver to determine eligibility for the intervention. Children with physician-diagnosed asthma and persistent symptoms in the past year according to national guidelines²² were eligible. Written informed consent was obtained from all primary caregivers before enrollment in this study.

From August 2006 to November 2006, we enrolled 226 of 314 eligible children (response rate, 72%). Each participating family received an extensive home visit to collect baseline data. We administered multiple surveys to collect demographic information, asthma symptom severity, medications, health care utilization, child behaviors, and care-giver factors. A saliva sample was collected from each child for cotinine measurement.

For this analysis we excluded children with an Autism diagnosis (including Autism, Asperger syndrome, and Pervasive Developmental Disorder, $n = 14$), and children with incomplete data ($n = 3$). We also excluded 9 children aged <4 years old because the behavior scale used for this analysis is not validated for children younger than 4. This final analytic sample includes both children with and without smokers in the home, for a total of 200 children. The University of Rochester's Institutional Review Board approved the study protocol.

Assessment of Asthma Severity

We assessed asthma severity during the baseline interviews by asking parents to report the number of days in the previous 14 days their child had daytime asthma symptoms and the number of days with nighttime asthma symptoms. Children with reported ≥ 5 days of daytime symptoms or ≥ 2 nights with asthma symptoms over 2 weeks were considered to have persistent asthma symptoms on the basis of national guidelines.²²

Assessment of Behavior

We assessed childhood behavior with the previously validated Behavior Problem Index (BPI).²³ The BPI was created by Peterson and Zill²³ using many of the same questions as the Achenbach Childhood Behavior Checklist.²⁴ This 32-item survey is used to assess behaviors during the previous 3 months for children 4 to 17 years of age, and 28 items are included in the survey for children 12 years and younger. Caregivers were asked to respond to statements of behavior by reporting whether each behavior was “not true,” “sometimes true,” or “often true” of their child. All positive responses (“sometimes true” and “often true”) were scored as 1 and summed to create a total behavior score (range, 0–28). A score >14 indicates significant behavior problems.²⁵ The internal consistency reliability of the full BPI scale was acceptable (Cronbach $\alpha = .90$). The BPI can also be divided into several subscales; externalizing behavior problems (18 items, $\alpha = .87$), internalizing behavior problems (10 items, $\alpha = .79$), anxious/depressed (5 items, $\alpha = .70$), antisocial (6 items, $\alpha = .61$), hyperactive (5 items, $\alpha = .76$), headstrong (5 items, $\alpha = .76$), peer conflict (3 items, $\alpha = .57$), and immature (4 items, $\alpha = .73$).

Assessment of ETS

We collected saliva samples from each child during the baseline assessment to determine the child’s level of cotinine, a metabolite of nicotine. Each child placed a sorbette (a wand with a small sponge on the end) or a salivate (cotton roll) under his or her tongue for 1 minute. Study staff collected 3 sorbettes or 1 salivate from each child to obtain sufficient saliva to be analyzed for cotinine. Salivary cotinine correlates well with parent report of smoke exposure,^{26,27} and it is used as a biomarker for many intervention studies for young children with asthma.^{28,29} All samples were measured with a standard enzyme-linked immunosorbent assay and reported in nanograms per milliliter. The lower limit of cotinine sensitivity is 0.05 ng/mL with a range of sensitivity from 0.06 to 200 ng/mL. Undetectable cotinine values were recorded as 0 ng/mL. ETS exposure was objectively assessed through the child’s cotinine level.

There is no clearly established level of cotinine that has been determined to be safe, and thus there is no clear cutoff for cotinine scores. Therefore, we divided the cotinine value at the arithmetic mean as a cutoff between low exposure and high exposure, and we also considered cotinine as a continuous variable. In addition, we asked parents about their self-reported smoking status (smoker vs non-smoker) and the number of smokers living in the child’s home (0 vs ≥ 1).

Assessment of Covariates

Covariates in this study consist of standard demographic variables for each child, including gender, race (white, African American, or other), ethnicity (Hispanic/not Hispanic), and child’s age. We also included Medicaid insurance (yes/no), prematurity (yes/no), and current asthma severity (intermittent vs persistent) as additional variables that may be related to ETS exposure and behavior. Additional variables included caregiver’s age (<30/ ≥ 30 years), caregiver’s education (less than high school/high school or more), parent depression, and stress. We evaluated parent depression with the Kessler Psychological Distress Scale,³⁰ a 10-item scale used to assess symptoms of depression and anxiety. We asked caregivers how frequently they experienced each item (eg, nervous, depressed) in the past 4 weeks (“none of the time” [score = 1] to “all of the time” [score = 5]). We summed scores from all items and higher scores indicate a higher risk of depression, anxiety, or both (range, 10–50). We then divided scores into 4 categories (well, mild, moderate, and severe psychological distress) on the basis of previously validated domains.³¹ We measured parent stress by using questions from the competence subscale of the Parenting Stress Index, with permission from the publisher, Psychological Assessment Resources, Inc.³² We included 5 items on a 5-point scale, and we summed scores for a total parent stress score (range, 5–25). Higher scores indicate increased parental stress.

Analysis

Analysis was performed by SPSS version 15.0 software (SPSS, Chicago, Ill). We conducted Student *t* tests to compare mean BPI scores for children with low (≤ 1.47 ng/mL) versus high (>1.47 ng/mL) cotinine scores. Multivariate regression analyses were conducted to determine the association between children's cotinine levels and BPI scores. Because of the nonnormal distribution of cotinine data, we used the natural log function to transform the data prior to the regression analyses. A 2-sided alpha $<.05$ was considered statistically significant. With our sample size of 200 (142 low cotinine vs 58 high cotinine), we anticipated having 80% power to detect a 2.8-point difference in BPI scores.

RESULTS

The demographic characteristics of the 200 children included in this study (mean age, 7.1 years) are shown in Table 1. Most children were male (56%), and 72% were enrolled in Medicaid. Sixty-five percent of the children were African American and 28% were Hispanic. More than half of the children lived with 1 or more smokers in the home. Ninety-five percent of children had detectable cotinine values ($>.05$ ng/mL); the mean cotinine level of all children was 1.47 ng/mL (range, 0–15.2 ng/mL). The average BPI score was 10.9 (range, 0–27). Overall, 30% of children had total BPI scores of >14 , which can be considered an indicator of significant behavior problems.²⁵

Table 1 also lists demographic characteristics separately for children with cotinine values above and below the mean. More children with cotinine values greater than the mean were insured by Medicaid and had a primary caregiver with less than a high school education. Cotinine values were consistent with the parent's report of smoking behaviors. Children with cotinine values above the mean were more likely to have more than one smoker living in the home and to have a primary caregiver who smokes compared with children with cotinine values less than the mean. There was no difference between the 2 groups regarding the prevalence of parent depression or stress. Of note, 44% of parents had a positive screen for depressive symptoms.

Table 2 compares cotinine values with parent-reported BPI scores. Children with cotinine values >1.47 ng/mL had significantly higher (worse) scores compared with children with lower cotinine values on the total BPI (12.5 vs 10.2, $P = .03$). In addition, children with cotinine values >1.47 ng/mL also scored higher on several subscales, including externalizing behaviors (9.0 vs 7.2, $P = .02$), antisocial (2.3 vs 1.7, $P = .02$), and immature (2.1 vs 1.6, $P = .04$). When we repeated the analysis using tertiles of cotinine, we found a similar trend of worsening total behavior scores with higher cotinine values (9.8, 10.8, 12.1, $P = \text{NS}$).

In individual multivariate models (Table 3) including demographic variables, parent depression and stress, asthma severity, and prematurity, log cotinine remained independently associated with externalizing behaviors ($R^2 = .30$, $\beta = 1.131$, $P = .04$), headstrong ($R^2 = .22$, $\beta = .448$, $P = .04$), and antisocial behavior ($R^2 = .25$, $\beta = .369$, $P = .04$). Higher cotinine scores were associated with worse behavior on these scales. Parent stress, parent depression, Medicaid, and prematurity also were independently associated with worse behavior scores among these children (results not shown).

DISCUSSION

This study assessed the relationship between ETS exposure and behavior problems among a group of inner-city children with persistent asthma. Overall, 30% of children scored >14 on the total BPI, indicating significant behavior problems that may warrant professional intervention.²⁵ This provides further evidence that children with asthma who live in an urban community have a high prevalence of behavior problems. High rates of ETS exposure also

were noted, with over half of the children living with one or more smokers in the home. We found that cotinine levels are associated with problem behaviors among this sample of urban children with asthma, even when controlling for pertinent and potentially confounding variables.

Specifically, we found that high levels of tobacco smoke exposure were associated with externalizing behaviors, including headstrong and antisocial behaviors, among urban children with asthma. Increased rates of externalizing behaviors have been found in previous samples of children exposed to tobacco smoke.^{18,19,33} For example, in a large longitudinal study that followed pregnant women and their children, the authors found strong associations between externalizing behavior in 4 to 6 year-olds and both prenatal and postnatal smoke exposure.¹⁹

Several studies have utilized the BPI as a parent-report measure to assess behavioral problems in children.^{6,18,34} For example, the BPI was administered in the 1988 National Health Interview on Child Health. A study that used these data compared behavior scores among children ages 5 to 17 years with asthma and children without chronic conditions.⁶ The authors found that children with significant asthma had a mean BPI score of 7.4 compared with 5.4 for children without a chronic condition. In comparison, the average BPI score for our sample of 4-to 10-year-old children with asthma was 10.9 (SD = 6.8). The higher scores found in our study may be caused in part by the different age ranges of children included in these studies and the different sampling frames.

We found a 2.3-point difference in total BPI score between children with low and high cotinine levels, representing approximately one-third of a standard deviation difference between groups. Although this difference is statistically significant, it is difficult to infer the clinical significance of this finding. The BPI score is a sum of positive responses for each behavior item, and thus the score can be interpreted as the mean number of behavioral concerns described by the parents. In our sample, children with a cotinine >1.47 ng/mL had, on average, 2.3 more items for which the parent reported that a described behavior problem was true for their child, compared with children with lower cotinine levels. This degree of difference is consistent with findings from a large nationally representative study where children exposed to maternal smoking had behavior scores 2.04 points higher than children with non-smoking mothers.¹⁸

Many factors may play a role in children's behavior. The families included in this study are from a poor inner-city community with high rates of public health insurance and parental depression. Family characteristics, environment, and stressful life events may have an effect on children's behavior and parental views of behavior. A strength of our study is that we are able to account for several items that may affect children's behavior. For example, previous studies have established associations between childhood behavior and parental stress³⁵ and depression.³⁶ Parents' education level and household income have also been linked with worse scores on the BPI.^{25,37} Furthermore, enrollment in Medicaid³⁸ and prematurity³⁹ have been shown to be associated with worse behavior in children later in life.

The high level of smoke exposure observed in this study is consistent with previous studies that have documented smoke exposure among children living in urban areas.¹⁴ In addition, as previously reported in the literature, we found that children were more likely to have a higher cotinine value if they were African American, insured by Medicaid, or had a parent with less than a high school education.^{10,12}

Nicotine, a key component of ETS, is a neuroteratogen with known adverse effects on the brain including fewer neurons, poor synaptic development, and poor neurobehavioral functioning.⁴⁰ Thus, the harm of prenatal smoking on the developing brain has been clearly established. Children exposed to ETS in childhood often are also exposed to prenatal maternal smoking, and therefore it is difficult to separate the 2 sources of exposure and subsequent developmental

outcomes in human studies. However, there is some evidence that postnatal ETS exposure in childhood, independent of prenatal exposure, may be hazardous to neurodevelopment. Studies in both rodents and rhesus monkeys have demonstrated changes in brain cell development when animals were only exposed to ETS postnatally.^{20,21} Although controlled trials in humans are not ethical, these animal studies indicate a potential mechanistic connection between postnatal ETS exposure and adverse neurobehavioral outcomes, thus strengthening the impetus for avoidance of ETS exposure in childhood.

Limitations

There are some limitations to this study. Behavior problems were assessed at one time point by one caregiver, and they were not confirmed by physicians or teachers or with subsequent assessments. In addition, because this is a cross-sectional study, we cannot establish a causal or directional relationship between ETS exposure and behavior problems in these children. All families were recruited from an inner-city community, and many of the families experience stressful lives that may contribute to both higher smoke exposure and parental views of behavior problems. For instance, we found a high rate of parent depression in this study, which could effect both child behavior and smoking status, as described in previous studies.^{36,41,42} Although we were able to control for several variables including parent depression and stress in our multivariate analysis, residual confounding is possible. Further, we did not have an exact measure of income in this study, and therefore we used enrollment in Medicaid as a proxy variable. Last, our sample included children with asthma from an urban school district, and these findings can only be generalized to a similar population.

Researchers have used various cutoff values to define levels of smoke exposure.²⁹ Because no clearly established level of cotinine has been determined to be safe, we divided the cotinine value at the mean and also considered cotinine as a continuous variable with consistent findings of increased behavior problems with higher levels of exposure. Further, when we reanalyzed the data using tertiles of cotinine levels, similar trends were seen. It is important to note that cotinine was detected in 95% of our sample. Furthermore, in children, salivary cotinine has a half-life of approximately 32 to 82 hours,⁴³ and therefore these values reveal ETS exposure for only a few days before collection. However, studies have suggested that a single cotinine measurement is reasonably consistent with routine exposure to ETS.^{43,44}

Implications

This study is unique in that it utilizes a brief and, easy to use behavior scale to assess childhood behavior problems. This scale could be used in pediatric clinics as a screening tool for potential behavior difficulties. Further, we used an objective measure of smoke exposure to assess the association between ETS and behavior problems among a group of vulnerable children.

Children with asthma and exposure to ETS represent a group at high risk for behavior problems. Although we cannot from this study determine whether reducing ETS exposure and improving asthma control would prevent behavior difficulties, it would be helpful for pediatric health care providers to be aware of this association and to provide appropriate screening and counseling in the primary care setting. In addition, the harmful effects of ETS on lung function and asthma morbidity are well described. Thus, tobacco counseling is needed as part of routine care for children with asthma.

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Table 1
Population Demographics by Low and High Cotinine Exposure

Demographic	Overall (N = 200)	Low Cotinine (mean ≤ 1.47 ng/ mL) (N = 142)	High Cotinine (mean > 1.47 ng/ mL) (N = 58)	P Value
Child gender, male, n (%)	112 (56)	84 (59)	28 (48)	.21
Child age, y, mean (SD)	7.1 (1.8)	7.01 (1.8)	7.3 (1.8)	.33
Race, n (%)				
White	18 (9)	9 (6)	9 (16)	.01
African American	130 (65)	89 (63)	41 (71)	
Other	52 (26)	44 (31)	8 (14)	
Hispanic, n (%)	55 (28)	41 (29)	14 (24)	.60
Medicaid insurance, n (%)	145 (72)	95 (67)	50 (86)	.005
Parent age < 30 years, n (%)	67 (34)	44 (31)	23 (40)	.33
Parent education less than high school, n (%)	69 (34)	42 (30)	27 (47)	.03
≥ 1 smoker in the home, n (%)	105 (52)	54 (38)	51 (88)	$< .001$
Caregiver smokes, n (%)	75 (38)	30 (21)	45 (78)	$< .001$
Child born premature, n (%)	23 (12)	15 (11)	8 (14)	.47
Persistent asthma, n (%)	122 (61)	88 (62)	34 (59)	.75
Parent depression, mild-severe mental disorder, n (%)	89 (44)	59 (42)	30 (52)	.21
Parent stress, mean (SD)	9.4 (3.3)	9.3 (3.3)	9.6 (3.2)	.60

SD indicates standard deviation.

Table 2

Overall and Subscales of Behavior Problem Index Scores by Low and High Cotinine Exposure

Behavior Problem [*]	Overall (N = 200)	Low Cotinine (mean ≤1.47 ng/ mL) (N = 142)	High Cotinine (mean >1.47 ng/ mL) (N = 58)	P Value
Total BPI, mean (SD)	10.9 (6.8)	10.2 (6.9)	12.5 (6.3)	.03
Externalizing, mean (SD)	7.8 (4.7)	7.2 (4.8)	9.0 (4.3)	.02
Internalizing, mean (SD)	3.1 (2.6)	2.9 (2.6)	3.6 (2.6)	.11
Anxious/depressed, mean (SD)	1.8 (1.5)	1.7 (1.4)	2.0 (1.5)	.18
Headstrong, mean (SD)	2.6 (1.8)	2.5 (1.8)	2.9 (1.6)	.17
Antisocial, mean (SD)	1.9 (1.6)	1.7 (1.6)	2.3 (1.5)	.02
Hyperactive, mean (SD)	2.2 (1.8)	2.1 (1.8)	2.6 (1.6)	.09
Peer conflict, mean (SD)	.58 (.86)	.53 (.86)	.69 (.86)	.23
Immature, mean (SD)	1.8 (1.4)	1.6 (1.4)	2.1 (1.4)	.04
Total BPI >14, n (%)	61 (30)	40 (28)	21 (36)	.31

* For values expressed as mean (SD), a high score indicates greater problem with behavior. BPI indicates Behavior Problem Index. SD indicates standard deviation.

Table 3

Linear Regression Models: Continuous Logarithmic Cotinine Predicting Behavior Problems*

Dependent Variable	β	R^2	P value
Total BPI	1.291	.30	.10
Externalizing	1.131	.29	.04
Internalizing	.159	.25	.61
Anxious/depressed	.165	.26	.34
Headstrong	.448	.22	.04
Antisocial	.369	.25	.04
Hyperactive	.194	.19	.37
Peer conflict	-.106	.20	.32
Immature	.221	.19	.21

* Data are adjusted for child age, gender, race, ethnicity, Medicaid, asthma severity, prematurity, parent depression, parent stress, parent age, parent education. BPI indicates Behavior Problem Index.